# The Yin & Yang of HIV

***When put to the test, conventional HIV/AIDS theory is at odds with the clinical evidence. Is "purified HIV" no more than a tangle of cellular debris?***

***Part 2 of 3***

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***The real purpose of scientific method is to make sure Nature hasn't misled you into thinking something you don't actually know... One logical slip and an entire scientific edifice comes tumbling down. One false deduction about the machine and you can get hung up indefinitely.***
— Robert Pirsig, *Zen and the Art of Motorcycle Maintenance*

## SOME SCIENTIFIC PROBLEMS WITH THE HIV THEORY

### • The theory vs the definition

The central premise of the HIV theory of AIDS is that there exists a unique retrovirus, transmissible via blood and sexual secretions, which induces specific antibodies and kills T4 cells whose relative absence then causes the appearance of approximately 30 diseases which constitute the clinical syndrome. The theory, however, is rendered completely contradictory by the official AIDS definition used clinically.

In Australia, an individual is diagnosed as having AIDS if he or she fulfills the criteria set out in the latest (1993) revision of the US "CDC surveillance case definition for AIDS".[107] (Other definitions in use around the world make scientific comparisons almost impossible. In Africa, AIDS is diagnosed on symptoms and without blood tests.[108]) Since from 1985 the Centers for Disease Control "accepts" HIV as the cause of AIDS, it should not be possible to diagnose AIDS by any means inconsistent with the HIV theory. However, even a cursory reading of the 1993 definition reveals AIDS can be diagnosed—with the imprimatur of the CDC—with Kaposi's sarcoma (which even Gallo[54] accepts is not caused by HIV), in the absence of immune deficiency, "without laboratory evidence of HIV infection" and, extraordinarily, "*in the presence of negative results for HIV infection*"[109] (italics ours).

### • Sexual transmission

HIV/AIDS is claimed to be bidirectionally sexually transmitted. Data to support this claim are based not upon microbial isolation and contact tracing, as is the orthodox practice for proving diseases are infectious and sexually transmitted, but on mostly retrospective studies of highly selected groups of individuals—including homosexual and bisexual men, heterosexual men and women including prostitutes—for antibodies in blood which react with certain proteins deemed "HIV-specific". Included in these studies are estimations of risk factors for the specific sexual practices of penile-insertive, vaginal, anal-receptive and oral-receptive intercourse.

### • Homosexual men

In 1984, Gallo and his colleagues showed that "Of eight different sexual acts, a positive HIV antibody test correlated only with receptive anal intercourse".[110] They also found that the more often a homosexual man had insertive anal intercourse, the less likely he was to become HIV-positive. This is incompatible with an infectious cause. In 1986, Gallo and his colleagues reported they "found no evidence that other forms of sexual activity contribute to the risk" of HIV seroconversion in homosexual men.[111]

In an extensive review of 25 studies of homosexual men reported in 1994 by Caceres and van Griensven, the authors concluded that "no or no consistent risk of the acquisition of HIV-1 infection has been reported regarding insertive intercourse".[112]

In the West, the largest and most judiciously conducted prospective epidemiological

studies, such as the Multicenter AIDS Cohort Study (MACS) of 4,954 gay men,[113] have proven beyond all reasonable doubt that in homosexual men the only significant sexual act related to becoming HIV-antibody-positive is receptive anal intercourse. Thus, in gay men, AIDS may be likened to the non-infectious condition, pregnancy. It is acquired by the passive partner but is not transmitted to the active partner.

Significantly, the MACS also showed that once a homosexual man becomes HIV-positive, progression to AIDS is further determined by the amount of passive anal intercourse sustained after "infection". This is contrary to all that is known about infectious diseases. Infection, not repeated infections, causes disease. Indeed, the Royal Australasian College of Surgeons (RACS) considers HIV-positive surgeons to be "infectious" and that they "should not perform invasive procedures or operations", but "they may provide these services to patients who have the same infection".[114]

### • Heterosexuals

The largest and best-conducted studies in heterosexuals, including the European Study Group,[115] showed that, for women, the only sexual practice leading to an increased risk of becoming HIV-antibody-positive is anal intercourse. The unidirectional transmission of "HIV" observed in OECD countries is supported by Nancy Padian's 10-year study of heterosexual couples (1986–1996). There were two parts to this study: one cross-sectional, the other prospective.

In the cross-sectional study, "The constant per-contact infectivity for male-to-female transmission was estimated to be 0.0009 [1 in 1,111]". The risk factors for the women were: (i) anal intercourse; (ii) having partners who acquired this infection through drug use (Padian says this means the women may also be IV drug users); (iii) the presence of STDs (antibodies to their causative agents may react in an "HIV" antibody test)[15, 20] Of the HIV-negative male partners of 82 HIV-positive female cases, only two became HIV-positive—but under circumstances that Padian considered ambiguous.

In the prospective study, starting in 1990, 175 HIV-discordant couples were followed for approximately 282 couple-years. At entry to the study, one third used condoms consistently and, in the six months prior to their last follow-up visit, 26 per cent of couples consistently failed to use condoms. There were no seroconversions after entry, including the 47 couples not using condoms consistently. Based on the 2 in 86 men who became HIV-positive in the early study, the risk to a non-infected male from his HIV-positive female partner was reported to be in the order of 1 in 9,000 per contact. From this statistic one can calculate that, on average, a male would need to have 6,000 sexual contacts with an infected female to achieve a 50 per cent chance of becoming HIV-positive. If sexual intercourse were to commence at age 20 and average three times weekly, this would occupy a lifetime.[57, 116]

**The "overwhelming" evidence from studies both in homosexual men and heterosexuals is that HIV/AIDS is not bidirectionally sexually transmitted. In the whole history of medicine there has never been such a phenomenon.**

### • Female Prostitutes

The notion that HIV is a virus which "does not discriminate" is also markedly inconsistent with the data obtained from studies of female prostitutes. Even if by some unknown means a sexually transmitted infectious agent found its way into the promiscuous portion of the gay male population in certain large cities in the United States in the late 1970s (as is widely accepted), and given the facts that prostitutes are frequented by bisexual men and that, at the very earliest, "safe" sexual practices date from 1985, one would have expected HIV/AIDS to have spread rapidly through prostitutes and thence to the general community. However, the prevalence of "HIV" antibodies amongst prostitutes is almost entirely confined to those who are drug users. Virtually all other prostitutes have not been, and are not becoming, HIV-positive.

In September 1985, 56 non-intravenous drug using (IVDU) prostitutes were tested "...in the rue Saint-Denis, the most notorious street in Paris for prostitution. More than a thousand prostitutes work in this area... These women, aged 18–60, have sexual intercourse 15–25 times daily and do not routinely use protection." None was positive.[118]

In Copenhagen, 101 non-IVDU prostitutes, a quarter of whom "suspected that up to one fifth of their clients were homosexual or bisexual", were tested during August–October 1985. The median numbers of sexual encounters per week was twenty. None was positive.[118]

In 1985, 132 prostitutes (and 55 non-prostitutes) who attended a Sydney STD clinic were tested for HIV antibodies. The average number of sexual partners (clients and lovers) in the previous month was 24.5. When an estimate was made to separate clients and lovers, the median number of sexual contacts per year rose from 175 to 450. The partners of only 14 prostitutes (11%) used condoms at all, and 49% of their partners used condoms in fewer than 20% of encounters. No women were HIV-positive.[119]

The same Australian clinic repeatedly tested an additional 491 prostitutes who attended between 1986 and 1988. Of 231 out of the 491 prostitutes surveyed, 19% "had bisexual non-paying partners and 21% had partners who injected drugs. Sixty-nine per cent always used condoms for vaginal intercourse with paying clients, but they were rarely used with non-paying partners. Condoms were rarely used by those clients and/or partners for the 18% of prostitutes practising anal intercourse." No women were HIV-positive.

At the time of this report, a decade into the AIDS era, the authors commented that "*there has been no documented case of a female prostitute in Australia becoming infected with HIV through sexual intercourse*" (italics ours). Yet, these investigators from the Sydney Sexual Health Centre concluded that "there are still many women working as prostitutes in Sydney who remain seriously at risk of HIV infection".[120]

In Spain, of 519 non-IVDU prostitutes tested between May 1989 and December 1990, only 12 (2.3%) had a positive test, which was "only slightly higher than that reported five years ago in similar surveys". Some prostitutes had as many as 600 partners a month, and the development of a positive antibody test was directly related to the practice of anal intercourse. The authors also noted that "a more striking and disappointing finding was the low proportion of prostitutes who used condoms at all times, despite the several mass-media AIDS prevention campaigns that have been carried out in Spain".[121]

Similar data from two Scottish studies,[122] the 1993 European

Working Group on HIV Infection in Female Prostitutes study,[123] and a 1994 report on 53,903 prostitutes working in the Philippines and tested between 1985 to 1992, confirm that non-IVDU prostitutes remain virtually devoid of HIV infection. For example, in the latter study, only 72 women (0.01%) were found to be HIV-positive.

In studies where there appears to be a high incidence of HIV amongst prostitutes, there are uncertainties that defy explanation. For example, although "HIV has been present in the commercial sex work networks in the Philippines and Indonesia for almost as long as it has been in Thailand and Cambodia", the prevalence of HIV in the former is 0.13% and 0.02% respectively and 18.8% and 40% in the latter.[124]

If these are accurate data, the discrepancy defies epidemiological explanation and has indeed baffled the experts, although the latter postulate "behavioural factors", such as one country's prostitutes and clients being considerably more or less sexually active than another. However, one could also pose another question. What are the "HIV" antibody tests actually measuring? Be that as it may, since 5,674 (44%) and 4,360 (34%) of the 12,785 Cambodian "HIV and AIDS Case Reports" till 31 December 1997 are listed as "Unknown" in gender and age respectively,[125] data collection, at least by the World Health Organization in Cambodia, must be regarded as problematic.

### • Contradictions

Why should HIV avoid non-drug-using prostitutes? If female prostitutes who do not use drugs do not become HIV-infected despite being "seriously at risk of HIV infection", what is the risk of infection to the majority of Australian women who are neither drug users nor prostitutes? According to data from the National Centre in HIV Epidemiology and Clinical Research, vanishingly little. A 1989 study testing 10,217 blood samples of newborn babies (unambiguous evidence of unprotected heterosexual intercourse) found no babies and thus, presumably, no mothers HIV-positive.[126] If such women remain non-infected, how do their non-drug-using, male heterosexual partners become infected with HIV?

According to Simon Wain-Hobson, a leading HIV expert from the Pasteur Institute, "a virus's job" is to spread. "If you don't spread, you're dead". The "overwhelming" evidence from studies both in homosexual men and heterosexuals is that HIV/AIDS is not bidirectionally sexually transmitted. In the whole history of medicine there has never been such a phenomenon. Since microbes rely on person-to-person spread for their survival, it is impossible to claim from epidemiological data that HIV/AIDS is an infectious, sexually transmitted disease. Indeed, Professor Stuart Brody, from the University of Tübingen, has argued that physicians ignore the actual heterosexual data and instead promote the politically correct idea that everyone is at risk. "Ideological knowledge about AIDS is far more likely to filter through society than scientific knowledge."[37]

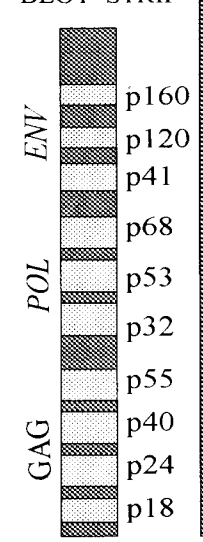
## THE DIAGNOSIS OF "HIV" INFECTION

### • The HIV antibody tests

There are two "HIV" antibody tests in common use: the ELISA and Western blot (WB). The ELISA causes a colour change when a mixture of "HIV" proteins reacts with antibodies in serum from a patient. In the WB, the "HIV" proteins are first separated along

## THE HIV WESTERN BLOT TEST

The HIV Western blot test consists of a thin nitrocellulose strip in which are embedded proteins claimed to be unique to HIV. Each protein is labelled with a "p" followed by its molecular weight in thousands. Serum is added to the strip and, if there are antibodies to a particular protein, this band will "light up". The HIV Western blot is not standardised, and thus, around the world, different combinations of bands are considered positive. Hence a positive test in one country is not positive in another. An African would not be positive in Australia. A person from the MACS would not be positive anywhere in the world, including Africa. Yet the HIV Western blot is considered to be highly specific and is considered synonymous with HIV infection.




| AFR | AUS | FDA | RCX | CDC 1 | CDC 2 | CON | GER | UK | FRA | MAC |
|---|---|---|---|---|---|---|---|---|---|---|
| ANY 2 | ANY 1 | ANY 1 | ANY 1 | p160/ p120 AND p41 | p160/ p120 OR p41 | p160/ p120 OR p41 | ANY 1 | ANY 1 | ALL 3 | 3 WEAK BANDS OR ANY STRONG BAND |
| | ANY 3 *GAG* OR *POL* | p32 AND p24 | ANY 1 AND ANY 1 | | AND | p32 OR p24 | ANY 1 *GAG* OR *POL* | p32 AND p24 | ANY 1 OR ANY 1 | |

Key: AFR = Africa;[1] AUS = Australia;[2] FDA = US Food and Drug Administration;[3] RCX = US Red Cross;[3] CDC = US Centers for Disease Control;[3] CON = US Consortium for Retrovirus Serology Standardization;[3] GER = Germany; UK = United Kingdom; FRA = France; MAC = US Multicenter AIDS Cohort Study 1983–1992

According to data presented in Lundberg et al.,[3] when the US FDA criteria are used to interpret the HIV Western blot, less than 50% of US AIDS patients are HIV-positive, whereas 10% of persons not at risk of AIDS are also HIV-positive by the same criteria.

Note: In February 1993, the US FDA relaxed its stringent criteria in order to "reduce the number of HIV-1 seroindeterminate Western blot interpretations"; that is, to increase the number of HIV-positive individuals.[4]

**Endnotes**

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2. Healy, D.S., Maskill, W.J., Howard, T.S. et al. (1992), "HIV-1 Western blot: development and assessment of testing to resolve indeterminate reactivity", *AIDS* 6:629-633.
3. Lundberg, G.D. (1988), "Serological Diagnosis of Human Immunodeficiency Virus Infection by Western Blot Testing", *JAMA* 260:674-679.
4. Kleinman, S., Busch, M.P., Hall, L. et al. (1998), "False-positive HIV-1 test results in a low-risk screening setting of voluntary blood donation", *JAMA* 280:1080-1083.

the length of a nitrocellulose strip. This enables individual reactions to the 10 or so "HIV" proteins to be visualised as a series of darkened "bands". The Western blot test is used to "confirm" repeatedly positive ELISAs because experts agree that the ELISA "overreacts"; that is, it is insufficiently specific.◊

Prior to 1987, one "HIV-specific" WB band was considered proof of HIV infection. However, since 15%–25% of healthy, no-risk individuals have "HIV-specific" WB bands,[127, 128] it became necessary to redefine a positive WB by adding extra and selecting particular bands, otherwise at least one in every seven people would be diagnosed as infected with HIV. (Notwithstanding, in the MACS, one band remained proof of HIV infection in homosexual men until 1990.[129]) On the other hand, although AIDS in Europe and the US began to decline in 1987,[130, 131] this trend was countered by the addition of more and more diseases and, most recently, mere laboratory abnormalities[132] to each revision (1985, 1987, 1993) of the first, 1982 CDC definition.

The net effect of these changes was to maintain a correlation between "HIV" antibodies and "AIDS" amongst the "risk" groups, while the risk of an HIV/AIDS diagnosis outside these groups remained slight. This was further accentuated by avoiding testing outside the risk groups. However, when such studies were performed amongst 89,547 anonymously tested blood specimens from 26 US hospital patients meticulously chosen to be at no risk of AIDS, 0.7%–21.7% of men and 0.0%–7.8% of women aged 25 to 44 years were found to be HIV-WB-positive.[133] (It is estimated that approximately 1% of men are homosexual. Also, at the five hospitals with the highest rates of HIV antibodies, one third of positive tests were in women. Yet men vastly outnumber women as AIDS patients.)

In addition, the US Consortium for Retrovirus Serology Standardization reported that 127 (10%) of 1,306 individuals at "low risk" for AIDS, including "specimens from blood donor centers", had a positive HIV antibody test by the "most stringent" US WB criteria.[127]

Thus the correlation of "HIV" antibodies with AIDS—which experts accept as the only *in vivo* proof that HIV causes AIDS—is not a statistic related to the natural, unbridled activity of a virus, but is instead a contrivance generated by mankind. Not only does correlation never prove causation, the artificiality of this particular "correlation" severely compromises its scientific analysis.

One of the most bizarre aspects of the HIV/AIDS theory is that different laboratories, institutions and countries define different sets of WB bands as a positive test (see chart on previous page). The global variation in interpretive criteria means that in Australia, for example, a positive test requires particular sets of four bands. In the USA, different sets of two or three suffice, which may or may not include the bands required in Australia. In Africa, only one designated set of two is required. Put simply, this means that the same person tested in three cities on the same day may or may not be HIV-infected.

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If the diagnosis of HIV infection were a game of poker, a flush would require five cards the same suit in one country, but only one or two elsewhere. A virus cannot behave in this manner, but according to the HIV test, which is claimed to have a specificity of 99.999%,[134] it does. As incomprehensible as this appears, further difficulties remain. For example, an Australian tested in Australia with one or two "HIV-specific" bands would not be reported as HIV-infected.[109] Clearly, however, there must be a reason why an uninfected individual, such as a healthy blood donor or military recruit, can possess any, even one, "HIV-specific" band. According to the experts, these bands are caused by cross-reacting, that is, "false", "non-HIV" antibodies which react with the "HIV" proteins. Thus it is axiomatic that an antibody which reacts with a particular protein is not necessarily an antibody which the immune system has generated specifically in response to that protein.

The Australian National HIV Reference Laboratory (NRL) concedes that "False reactivity may be to one or more [HIV] protein bands and is common (20%–25% of anti-HIV-negative blood

**Endnotes**

◊ In most countries, including Australia, individuals with two positive ELISAs have HIV infection "confirmed" by performing a Western blot test. However, this testing algorithm selects individuals who have a higher rate of cross-reacting antibodies and are therefore more likely to react in the Western blot test. (This is analogous to determining the number of heart attacks in the community by performing ECGs only on patients with chest pain—an experiment which grossly underestimates the real number because many heart attacks are "silent".) On the other hand, England and Wales do not use the HIV WB to "confirm" reactive HIV ELISAs because Dr Philip Mortimer, Director of the UK Public Health Laboratory Service, claims that "truly positive" antibodies are "easily" detected "because these are reactive in all ["methodologically different machine-read" ELISA] assays".[181] (This reasoning is analogous to performing a chest X-ray with several machines and claiming a suspicious abnormality is lung cancer because the appearances are repeatedly the same.) Asked at the 1998 Geneva AIDS Conference to comment on the UK dropping the Western blot test, Gallo remarked: "Well, the bulk of the world uses it. If some technology comes across better, I'd be the first to say 'do it'. I mean, obviously, the Western blot's a valuable test as defining the proteins that you have antibodies to. Everybody uses it experimentally and most people use it around the world... Britain doesn't use it. Maybe there are two countries that have found a better way. God bless them. Okay?"

◊◊ In 1997, the Perth group attempted a second time to engage the Royal Australasian College of Surgeons (RACS) in debating the HIV/AIDS controversy by submitting a paper entitled "A critical analysis of the evidence for the isolation of HIV" (see website <www.virusmyth.com/aids/data/epappraisal.htm>). It is RACS editorial policy to "welcome personal views of surgeons on a variety of topics" and to publish papers on "current and controversial issues". Although both reviewers accepted the bulk of the scientific arguments and found the paper "interesting reading", they advised against publication because, in their view, an analysis of evidence for the isolation of HIV was of "no real relevance...to a surgical audience" or "would be of little interest or use to the majority of readers of the *Australian and New Zealand Journal of Surgery*".

◊◊◊ Of the cumulative 7,766 Australian AIDS cases to date, 387 (5%) are reported in the "Heterosexual contact" exposure category. However, 22 of these qualify on the basis of "Sex with injecting drug user", "Sex with bisexual male", "From high prevalence country" (where heterosexual spread is deemed dominant), "Sex with HIV-infected person, exposure not specified", or "Not further specified".[177] Thus, injecting drug use, anal intercourse in women, the presumption of any form of sexual intercourse, and lack of sufficient data, question the mode of acquiring HIV infection in at least 330 (85% of) individuals listed in this exposure category.

donors [will] exhibit one or more bands on a WB)".[128] However, Eleopulos argues that if "non-HIV" antibodies cause "one or more protein bands", then why are they not able to cause four or five? Or all ten? On what basis do experts assert which antibodies are "false" and which are "true"? Or, how do the same three bands, caused by "false", non-"HIV" antibodies, become "true" when accompanied by one extra? On what basis do experts assert there are any "true" HIV antibodies? If the Australian traveller were to be tested in the USA, where two or three bands are sufficient to diagnose HIV infection, are his antibodies "false" in Australia but "true" as his aeroplane touches down in Los Angeles?

In 1994, one of us (VFT) wrote to the *Medical Journal of Australia*, seeking justification of both the Australian criteria for a positive Western blot test and the global variability.[28] The response by Dr Elizabeth Dax of the NRL[135] did not answer either question, and subsequent correspondence failed to pass the editorial staff at the same journal. When the same questions were later put via the offices of Senator Chris Ellison, the first question was again unanswered, and the widely different criteria between Australia and Africa were justified on the basis that, in Africa, "comparatively false reactivity is far less common [than in Australia] so that interpretation criteria to define [true] positivity may be less strict".[128] However, no scientist can make such a claim without data.

**If "HIV" antibodies indeed prove that one third of heterosexual adults in certain central and eastern African countries are infected with HIV, "life in these countries must be one endless orgy".**

All antibody tests are subject to the vagaries of cross-reactions, and the only way to calculate the incidences of "true" and "false" antibodies is to scrutinise reactions against what the test is purportedly meant to measure, that is, against HIV itself.

HIV isolation is the only "gold standard" by which the specificity of the antibodies can be determined, and this must be evaluated before the test is introduced into clinical practice.

However, despite the WB test being in widespread use and "a stalwart"[135] of HIV testing, these data have never been reported by the NRL or any other laboratory. Even without such evidence—since (a) the NRL concedes that cross-reacting antibodies cause misleading reactions in the WB in one quarter of healthy Australians, and (b) unlike Australians, Africans (similar to the AIDS risk groups) are exposed to a multitude of infectious agents producing myriad antibodies, each capable of cross-reactions—"false reactivity" will be much higher in Africa where the WB criteria should be the most stringent. If "HIV" antibodies indeed prove that one third of heterosexual adults in certain central and eastern African countries are infected with HIV, "life in these countries must be one endless orgy".[39]

If the proteins used in the HIV ELISA and WB tests are unique constituents of an exogenous retrovirus, and if such a virus induces specific antibodies, we would never expect to find such antibodies in the absence of HIV. Yet, in addition to the circumstances above, there are numerous others where antibodies which react with the "HIV-specific" proteins arise where HIV/AIDS experts concede there is no HIV. These include healthy mice injected with lymphocytes of similar mice[136] or bacterial extracts (V. Colizzi et al., personal communication); following the transfusions of HIV-free blood[137] or a person's own irradiated blood[138]; and 72 out of 144 dogs tested at a veterinary clinic in Davis, California, USA.[139] In addition, antibodies to the microbes which cause the fungal and mycobacterial diseases affecting 90% of AIDS patients react with the "HIV-specific" proteins.[20, 140]

This year it was reported that 35% of patients with primary biliary cirrhosis, 39% of patients with other biliary disorders, 29% of


**Endnotes (continued)**

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those with lupus, 60% of patients with hepatitis B, 35% with hepatitis C—all non-HIV, non-AIDS diseases—have antibodies to the "HIV" p24 "core" protein.[141] Until 1990, an unknown number of the 4,954 homosexual men in the MACS were diagnosed HIV-infected on the basis of an antibody to the "HIV-specific" p24 protein, that is, with one WB band. Why do not all similar tests prove infection with HIV? Why are gay men with a single p24 band infected with a deadly virus, while biliary and liver disease patients with the same band are not? Why were the criteria for diagnosing HIV infection set less rigorously for homosexual men and Africans? And if HIV antibodies are specific and HIV infection is "for life", why do reformed drug addicts, leading healthy lives, lose their HIV antibodies?[142]

Although all HIV experts accept cross-reactivity in HIV antibody testing, in 1993 the New South Wales Department of Health interpreted the discovery of "HIV" antibodies in four women as "compelling evidence" for transmission of HIV from a homosexual man during the course of minor, office surgery in 1989.[143] However, there was no proof that the man was HIV-infected at the time of surgery, or that any of the four women were operated on after the man.

This report remains the only one of its kind in the world, and it immediately led to the establishment of a special committee of the Royal Australasian College of Surgeons which wrote to all College Fellows, inviting submissions upon the matter. But, rather than seizing upon the rarity of the event and following advice urging a formal scientific enquiry into whether "HIV" antibodies are caused by infection with a retrovirus,[144] the College accepted these data as proof of cross-infection but concluded, "The mode of transmission is unknown".[114] ◊◊

Unlike HIV/AIDS experts, who claim the specificity of the HIV antibody test is 99.999%, one manufacturer of HIV antibody tests states in the package insert: "At present there is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood. Therefore sensitivity was computed on the clinical diagnosis of AIDS, and specificity based on random [healthy blood] donors..."[145] The latter were chosen as *de facto* non-HIV-infected for the purposes of determining how many tests are false positives. However, by this "reasoning", since the majority of HIV-positive individuals are healthy, they cannot be infected. Thus the WHO[146] predictions of a global pandemic are patently untrue.

**One manufacturer of HIV antibody tests states in the package insert: "At present there is no recognized standard for establishing the presence or absence of HIV-1 antibody in human blood..."**

**Editor's Notes:**

• In Part 3, concluding this series (NEXUS 6/06, Oct-Nov 1999), the authors continue their "HIV" exposé, questioning the scientific "proof" at the heart of mainstream AIDS research and discussing the "dissident" viewpoint in terms of politics and public health policy.

• Some of the endnote references in Part 2 of this article are to be found in Part 1, published last issue (NEXUS 6/03, June-July 1999).

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